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Attorneys for Plaintiffs

COLUMBIA RIVER UNITED,

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IN THE UNITED STATES DISTRICT COURT FOR THE WESTERN DISTRICT OF WASHINGTON

DIOXIN/ORGANOCHLORINE CENTER, and )

Plaintiffs,

DANA A. RASMUSSEN, et al.,

Defendants.

No. C91-1442-C

DECLARATION OF IAN CHRISTOPHER THOMAS NISBET

I, IAN CHRISTOPHER THOMAS NISBET, declare as follows:

- My name is Ian Christopher Thomas Nisbet I am currently employed as President of I.C.T. Nisbet & Company, a scientific consulting firm. My business address is 72 Codman Road, Lincoln, Massachusetts 01773.
- I hold a Ph.D. degree from the University of Cambridge. I am a professional environmental scientist. A copy of my Curriculum vitae is attached hereto as Exhibit A and is incorporated herein by reference.
- Since 1969, my principal professional work has involved assessment of the distribution and effects of toxic chemicals in the environment. I have specialized in assessment

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of chlorinated hydrocarbons, including the chemicals known as polychlorinated dibenzo-p-dioxins ("PCDDs" or "dioxins"). I have conducted numerous risk assessments for these compounds, including assessments of their likely effects on both human health and the environment, including wildlife. Among other things, I have conducted health and environmental risk assessments for seven dioxin-contaminated sites on behalf of the U.S. Environmental Protection Agency (USEPA). I am currently a consultant to the U.S. National Research Council on risk assessment methodology, including methodology for ecological risk assessment.

- assessments of the effects of chlorinated hydrocarbons on fisheating birds since 1969. I have published a number of scientific papers on this subject, including two review articles [1, 2]. In 1988, I presented an analysis of the effects of chlorinated hydrocarbons on the Bald Eagle at an international conference and my paper was published in the proceedings of that conference in 1989 [3]. In 1990, I served as a participant and as conference summarizer at an expert consultation meeting on the Bald Eagle, convened by the International Joint Commission [4].
- 5. I have reviewed a document entitled <u>Total Maximum</u>

  <u>Daily Loading (TMDL) to Limit Discharges of 2,3,7,8-TCDD (Dioxin)</u>

  <u>to the Columbia River Basin</u> (the "TMDL Document") and <u>Responses</u>

  <u>to Comments Received Concerning the Proposed Dioxin TMDL for the</u>

  <u>Columbia River Basin</u> issued by USEPA on 25 February, 1991 [5]. I

  have also reviewed other related documents, including USEPA's

Ambient Water Quality Criteria Document for 2,3,7,8
tetrachlorodibenzo-p-dioxin (the "AWQC Document") [6], and

USEPA's 1990 Work Plan for Investigation of Toxins in the

Columbia River Basin (the "1990 Work Plan") [7]. I have also

reviewed scientific studies of the behavior of PCDDs and other

chlorinated hydrocarbons in the environment, and of the effects

of these Chemicals on fish-eating birds, including the Bald Eagle

[1-4, 8-19].

- 6. In my professional opinion, the TMDL Document is scientifically deficient and the TMDL promulgated therein would be inadequate to protect the natural environment of the Columbia River Basin and Bald Eagles living there. The principal bases for this opinion are given in paragraphs 7-15 following.
- 7. The TMDL Document addresses only one of the many toxic chemicals found in the Columbia River Basin. This chemical -- 2,3,7,8-tetrachlorodibenzo-p-dioxin ("2,3,7,8-TCDD") -- is one of the class of chemicals known as polychlorinated dibenzo-p-dioxins ("PCDDs" or "dioxins"). PCDDs, along with other related compounds (e.g., polychlorinated dibenzofurans or PCDFs and polychlorinated biphenyls or PCBs), share the property known as "bioconcentration" -- that is, they accumulate in the tissues of aquatic organisms, including fish, to concentrations much higher than those in the ambient water to which the organisms are exposed. In a process known as "biomagnification," these compounds are further concentrated into the tissues of animals that consume the contaminated organisms, including fish-eating birds such as Bald Eagles.

8. These compounds are known to produce a variety of toxic effects in mammals and birds, including the induction of aryl hydrocarbon hydroxylase (AHH), inhibition of weight gain, skin lesions, edema, thymic atrophy, immune system disfunction, reproductive impairment including developmental effects, endocrine dysfunction and neurological impairment. Many of these toxic effects of 2,3,7,8-TCDD and dioxin-like compounds have been observed in populations of wild mammals or birds [10-13, 15-16]. Fish-eating birds accumulate these compounds in their body fat when they consume contaminated prey. These compounds can then be passed, along with fat, to the egg at the time of its formation.

- 9. In its TMDL Document, USEPA attempted to determine the amount of 2,3,7,8-TCDD that could be discharged to the Columbia River without violating state water quality standards [5, Appendix A]. These state standards were themselves based on USEPA's Ambient Water Quality Criterion (AWQC) for 2,3,7,8-TCDD of 0.013 parts per quadrillion (ppq). USEPA derived this value by considering possible cancer risks to human consumers of fish from contaminated waters [6]. However, at the time when the AWQC Document was prepared, there was insufficient information to establish a water quality criterion for protection of aquatic organisms [6]. In fact, the AWQC Document stated explicitly that the criterion was not meant to protect all species [6], and the AWQC Document did not consider fish-eating mammals or birds in any way.
  - 10. It is not scientifically justifiable merely to assume that the AWQC of 0.013 ppq would protect wildlife, for at

least two reasons. For one, wildlife, especially fish-eating 1 mammals and birds, are much more highly exposed to water-borne 2 contaminants than are humans. For example, most or all of the 3 diets of fish-eating wildlife species consist of fish, whereas fish comprise less than one percent of the diet of the average 5 person in the United States. Second, some wildlife species, 6 including the mink [10] and certain fish-eating birds [10-11], 7 are especially susceptible to the toxic effects of PCDDs; these 8 species may well be more susceptible than humans, even if they 9 were exposed to the same doses, which they are not. 10 reasons, there is no basis whatsoever for concluding that the 11 criterion of 0.013 ppq would necessarily be protective of 12 wildlife species, such as fish-eating Bald Eagles. 13 14 15 16

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11. The AWQC Document [6] was issued in 1984 and much of the information on which it was based has been superseded by new scientific information. In particular, much more information is now available on the bioconcentration and biomagnification of 2,3,7,8-TCDD in fish and wildlife species, and on its toxic effects in fish, birds, and mammals. Based on this new information, EPA could have estimated the potential for reproductive and developmental damage to wildlife species, such as Bald Eagles (see paragraph 15 below).

Another serious deficiency of the AWQC Document, as the basis for present-day standard-setting, is its treatment of bioconcentration of 2,3,7,8-TCDD in fish. Based on information available in 1984, the AWQC Document adopted a value of 5,000 as an estimate of the Bioconcentration Factor (BCF).

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The BCF is the factor by which the concentration of a chemical in water can be multiplied to estimate the average concentration in This average concentration is then used to calculate the exposure of consumers of the fish, and hence to estimate risks to these consumers. Although the AWQC Document adopted the value of 5,000 for the BCF, it acknowledged that much higher values (up to 900,000) had been predicted for the BCF in published scientific studies, so that concentrations of 2,3,7,8-TCDD in fish might be Work reported since 1984 has underestimated [6, p.B-10]. confirmed that the value of 5,000 is much too small [17]. Hence, the actual exposures and risks calculated in the AWQC Document were much too small. In other words, the 1984 criterion was not adequately protective of human consumers, let alone species which consume large quantities of fish in their diets, such as Bald Eagles.

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the columbia River system are already contaminated with 2,3,7,8-TCDD to hazardous levels [5, p. 3-2], it fails to take account of this existing contamination in calculating the daily loading that it would permit in the future. Based on the discussion in Appendix B of the TMDL Document, the existing contamination of the fish must be derived from discharges of 2,3,7,8-TCDD into the river in the past, which would have resulted in retention of some of the discharged chemical in the sediments, followed by uptake into the fish. Any continued discharge into the system in the future will augment the existing contamination and may result in an increase in the levels of 2,3,7,8-TCDD in the fish above the

present, already unacceptable, levels of contamination. The TMDL Document fails to take into account this existing contamination in calculating the daily loading (i.e., the TMDL) that it would permit in the future, however. As pointed out in paragraph 15, below, loadings of 2,3,7,8-TCDD in the Columbia River System are already sufficiently high to impair the reproduction of Bald Eagles. Additional loadings will augment the exposure of the Bald Eagles and will further contribute to their reproductive impairment.

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The TMDL does not reflect current scientific knowledge or even customary EPA procedures in regard to multichemical contamination. The TMDL Document considers and establishes discharge limitations for only one chemical --2,3,7,8-TCDD. This chemical is only one of the family of chemicals known as PCDDs, and it occurs in industrial discharges (including pulp mill discharges) and in the environment in conjunction with other PCDDs and with other related chemicals [18]. It is well-established scientifically that many of these chemicals exert their toxicity by the same receptor-mediated mechanism and that the effects of the various individual chemicals are approximately additive [11, 19-22]. Hence, complex mixtures of PCDDs, PCDFs, and related chemicals that occur in the environment are more toxic -- often much more toxic -- than would be predicted from the concentration of 2,3,7,8-TCDD alone. and others have long recognized this and accordingly have developed procedures for calculating the toxic potential of mixtures of dioxin-like compounds, using toxic equivalency

factors ("TEFs") [11, 19, 21]. However, USEPA has failed to apply its own procedure, or any other procedure, to the Columbia River Basin, considering only one chemical in the TMDL Document [5]. This failure to consider the effects of chemicals other than 2,3,7,8-TCDD means that USEPA has understated the risks to Bald Eagles and their environment, and overstated the degree of protection. Thus, any claim that USEPA's TMDL would protect Bald Eagles or their environment is misleading and incorrect.

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Although many fish and wildlife species are 15. exposed to PCDDs and related compounds in the Columbia River Basin, the Bald Eagle is at particular risk of high exposure. Columbia River Bald Eagles not only consume large quantities of fish (approximately 71% of their diets) [9], but also consume fish-eating birds which themselves concentrate PCDDs and related compounds to levels higher than those in the fish [9, 23]. Bald Eagles are thus subject to greater exposure to these chemicals than are other species which eat only fish. Studies in the Great Lakes have shown that Bald Eagles accumulate extremely high levels of PCDDS and related compounds in their tissues [19, 23]. Bald Eagles along the Lower Columbia River already suffer from impaired reproduction, associated with exposure to chlorinated hydrocarbons [9]. Although the relative roles of 2,3,7,8-TCDD and other compounds in reproductive impairment of the Columbia River Eagles have not yet been studied [9], there is good reason to conclude that these eagles are exposed to hazardous levels of 2,3,7,8-TCDD. Data from USEPA's National Bioaccumulation Study indicate that fish from the Columbia River Basin generally

contain 2,3,7,8-TCDD at concentrations in the range of 1-8 parts 1 per trillion (ppt) [7, Table 2]. Studies in other fish-eating 2 bird species have indicated that biomagnification factors 3 (concentration in bird tissue divided by concentration in fish) 4 for 2,3,7,8-TCDD are about 37 for eggs [11] and 32 for whole 5 Thus, the concentrations of 2,3,7,8-TCDD in the bodies [14]. 6 bodies and eggs of fish-eating birds in the Columbia River Basin 7 would be expected to be in the range 30-300 ppt. These are well 8 into the ranges associated with toxic effects in other species 9 [12, 24], even without considering the likely greater exposure of 10 Bald Eagles resulting from their consumption of fish-eating birds 11 as well as fish. Thus, it is probable that 2,3,7,8-TCDD at 12 existing levels of contamination is already contributing to 13 reproductive impairment in Bald Eagles in the Columbia River 14 Basin. Because the toxic mechanisms by which 2,3,7,8-TCDD and 15 other dioxin-like chlorinated hydrocarbons act are similar or 16 identical, the effects of 2,3,7,8-TCDD would augment the effects -17. of these other chemicals. Continued release of 2,3,7,8-TCDD into 18 the system would further augment the exposure of the eagles, as 19 discussed in paragraph 13 above, and hence adverse effects, such 20 as reproductive impairment, would also be augmented. 21

professional opinion that the TMDL promulgated does not reflect current scientific knowledge and would not protect Columbia River Bald Eagles or their environment. Continued discharge of 2,3,7,8-TCDD (in conjunction with discharge of related compounds and pre-existing contaminant levels) would probably cause adverse

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effects on bald eagles and other aquatic wildlife.

I declare under penalty of perjury that the foregoing is true and correct to the best of my knowledge. Executed this <a href="https://dx.nowledge.com/like/day">https://dx.nowledge.com/like/day</a> of November, 1991, in Lincoln, Massachusetts.

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. JAN CHRISTOPHER THOMAS NISBET

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EXHIBIT A

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